Modern Concepts of Cardiovascular Disease

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IMMERSION FOOT

I. Introduction

Trench foot and its seagoing counterpart, immersion foot, are both caused by prolonged exposure of the dependent lower extremities to cold and moisture. Wet cold is far more difficult to deal with than severer degrees of dry frost, as was found by Baron Larrey in the retreat of the Napoleonic armies from Moscow and by our medical officers on duty with the troops stationed in Attu and Kiska. This article, being limited to the local circulatory effects of immersion, is therefore confined to experiences with survivors of torpedoed vessels who have developed the syndrome commonly called immersion foot during a prolonged period adrift in the cold water of the North Atlantic.[1]

II. Pathological Physiology

In a crowded lifeboat or Carley raft the crew is forced to sit up with their wet feet dependent and immobile. This alone is sufficient to produce oedema, and if starvation is an added factor, so that the plasma proteins are reduced, the oedema will become pronounced, even in the absence of cold. No lifeboat or raft is dry, and boots are not issued that will keep the feet both dry and warm. Occasionally when a man has been forced to kneel for prolonged periods on the floorboards of a wet boat, or to sit on the bottom of a rubber raft, the same condition has involved the knees or the buttocks.

Living tissue will not freeze above the freezing-point of sea water, but at temperatures from this point up to about 50° (F.) the feet gradually become chilled, numb, and swollen. These changes take place in the course of a few hours to several days, depending on the degree of chilling. When the exposure has been severe, blisters develop. The condition at sea differs from that seen in troops campaigning in cold mud, simply because men in a crowded lifeboat do not have the opportunity to walk on their anaesthetic feet and to rupture their blisters. When this occurs infection is likely to follow and end in moist gangrene. This complication is fortunately rare, and amputation has been performed only twice in approximately 100 cases that I have had the opportunity to examine.

At the time of rescue these feet are cadaveric in color, swollen, and blistered. Massive gangrene from the ankle down may appear imminent, yet recovery is usually complete. It takes place in two stages, an early hyperaemic stage, and a later period of fibrosis. If the exposure has been severe and prolonged, pain may be severe in both and a cause of prolonged incapacity.

The hyperaemic phase develops as soon as the legs become warm and circulation recovers. This vasodilator response is a sterile inflammatory reaction caused by the absorption of damaged tissue. There are bounding pulsations in the deep vessels and heat, radiating to the surface, makes the skin fever-hot. But the circulation of the superficial tissues, which have been more thoroughly penetrated by the cold, may be profoundly impaired. Evidences of this are the petechial haemorrhages caused by ruptured capillaries, and the congestion of the feet in the dependent position. The combination of heat, which raises the metabolic demands of the cells for oxygen, and an inadequate superficial circulation results in cutaneous anoxia and burning pain as soon as the nerves recover. Recovery of sensation takes place rapidly over the proximal parts of the feet, but in the severer cases the toes may remain anaesthetic for prolonged periods.

In the course of one to four weeks the hyperaemia subsides and the circulation returns to normal. The blebs absorb and the skin peels off with a certain amount of scarring. The milder cases are then fit to return to duty, but in those with more severe damage cutaneous sensory recovery is slow, movement of the toes limited, and walking made difficult by pain. No satisfactory explanation for the rigid, weak feet and pain had been given until recently. This is probably two-fold. Ungley[2] has suggested that the prolonged discomfort is due to incomplete recovery of the sensory nerves, as hyperpathia (interpretation of all forms of sensory stimuli as pain) is a common phenomenon in cases of partial nerve injury. Another important causative factor of persistent disability is the fibrosis which follows prolonged chilling short of actual frostbite, the extent of which has not heretofore been appreciated. During the past winter Lt. Commander Shields Warren and I[3] examined a series of biopsies of skin and superficial muscle from the feet of 6 men who were still incapacitated several months after their exposure. These biopsies show atrophy and thinning of the epidermis, with intense fibrosis and deposition of collagen around the nerve endings and subcutaneous blood vessels. The muscles are also infiltrated and their fibrils separated by a network of scar tissue. The extent of this reaction depends on the initial exudation of fibrin and tissue anoxia. It is the logical explanation for the late pain, rigidity, and weakness of the feet, a condition which tends to improve spontaneously after six to eight months, the time at which collagen ceases to contract.

A final point to remember in the pathogenesis of these swollen, painful feet after prolonged periods adrift is the role of malnutrition and avitaminosis. I have seen extreme swelling, numbness, and pain develop after two weeks of dehydration and starvation with exposure in the warm waters of the Gulf Stream. This was not caused by tissue injury from cold, but by hypoproteinaemia and avitaminosis (particularly by depletion of the B complex).[4]

III. Treatment

When men are rescued at sea with chilled, numb, swollen, and blistered lower legs, they must be kept off their feet and the skin protected against rupture of the blebs and the formation of pressure sores in the weight-bearing areas of anaesthetic skin. Once streptococcal infection starts, moist gangrene and loss of the leg are likely to follow. First aid should therefore begin by having the patients carried below and kept in bed. Entirely different forms of treatment are required for the general effects of cold and the local damage from exposure. The body should be

warmed externally by covering with blankets and by hot water bottles; internally by drinking hot soup, coffee, or tea. The men should be given nutritious, easily digested food with a high protein-vitamin content as soon as it can be tolerated. In the sickest survivors vitality may have been so reduced by cold and starvation that emergency treatment for shock is needed. Transfusion of plasma will then play a double role by restoring depleted fluid volume and by combatting hypoproteinaemia.

It is vitally important to keep the injured feet cool, while the body of the chilled patient is warmed. This is best accomplished by exposing the legs below the knees to a cool room temperature and directing a blast of air from an electric fan or blower over them. Never rub these feet or paint the blistered areas with strong disinfectants. These areas should be handled like a second degree burn with aseptic precautions (including masking of the dresser's nose and mouth), sulfonamide therapy, and a "booster" dose of tetanus toxoid. Elevating and cooling the legs will help drain the oedema fluid and cause the blisters to shrink. During the period of sterile inflammation the hyperaemia of the deeper tissues, which are always less damaged than the skin by cold, will cause the surface temperature to rise to above 90°. Unless this excessive heat is reduced, cyanosis will increase, blisters swell, and pain become acute. The explanation of this is that warming increases the metabolic demand for oxygen on the part of the cutaneous cells to a greater extent than can be met by the supply of blood through the injured subcutaneous vessels. Anoxia therefore ensues with throbbing pain, increased extravasation of fluid, and necrosis of the skin.

After arrival in port and transfer to a hospital the same local treatment should be continued for a week or more, until the hyperaemia due to absorption of damaged tissue has subsided and circulation through the thrombosed subcutaneous vascular bed has recovered. The greatest advance in the treatment of the acute hyperaemic stage has been the application of continuous cooling by Webster, Woolhouse, and Johnston.[5] In general, this should be pushed to the point where pain is relieved. The ideal cutaneous temperature level is between 75° and 70°. Cooling lowers the metabolic need for oxygen to a point at which sufficient quantities can be supplied through the injured capillary bed. When the feet are cooled the appearance of the skin improves, blisters shrink, and pain is relieved. In the severest cases cooling with room air is insufficient and treatment with ice-bags may be necessary. When ice-bags are to be used, sterile pledgets of cotton are placed between the toes and the whole foot covered with a sterile towel. Thoroughly dried ice-bags are then placed around each foot over the towel and the whole encased in an oiled silk bag, around which in turn are wrapped thick layers of cotton waste or other insulating material. A rubber pillow-case, loosely tied below the knee, forms a satisfactory outer covering. At first two to four ice-bags will be necessary to cool the foot to the optimum temperature of 75° to 70°. Ordinarily it is sufficient to change them every four hours, and at this time the toes should be palpated to make sure that they are not too severely chilled. It may be necessary to carry out this treatment for several days to two weeks, but as hyperaemia subsides less ice is required and finally exposure to cool air alone will suffice.

When the products of tissue damage have been absorbed circulation returns to normal, blebs absorb, and the skin desquamates. In the milder cases the patient may be fit to return to duty within two weeks, but in the more severe disability may last for months. This is due to fibrosis of the skin and underlying tissues. Intermittent shooting pain at rest and increasing discomfort on walking are the chief complaints; the feet are weak, and the toes rigid. Physiotherapy and orthopedic exercises are helpful in restoring power to the intrinsic muscles and joints of the feet, but often no treatment is really effective until the infiltrating scar tissue ceases to contract. This occurs at the end of six to eight months, at which time the worst cases I have seen have become free from pain and able to return to work, even if only to light duty. In some of the most severe cases reported by Ungley the pain is still present at the end of three years. It would be a real saving in manpower for the Navy and Merchant Marine if a method could be found to expedite recovery during this late period of fibrosis. Sympathectomy has been proposed and warrants extensive trial and critical observation. Although this operation is quite illogical in the early hyperaemic stage, it may well expedite healing and relieve pain in the chronic stage of fibrosis by improving the impaired superficial circulation. It is hoped that this problem can be settled during the coming winter.

Much can be done to reduce the severity of exposure from immersion, and it is equally important to avoid further injury after rescue from improper first aid measures. With this end in view the National Research Council is preparing simple sheets of instruction for the Medical Corps of the Army and Navy, and also one for torpedoed seamen. If infection and added fibrosis from too rapid thawing can be prevented or reduced, the serious complications and prolonged disability which result from exposure due to immersion will be greatly decreased.

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